Conclusions: Aggravated injury of coronary NR after myocardial I/R in type 2 diabetic rats is associated with lower serum adiponectin levels, and exogenous administration of adiponectin could effectively alleviate NR injury in type 2 diabetic rats and improve cardiac function as well.

GW25-e3563

Role of late sodium current in ventricular arrhythmias caused by increased intracellular calcium concentration

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Objectives: Increase in intracellular calcium concentration is associated with prolongation of action potential duration (APD) and polymorphic ventricular tachycardia (PVT). Recent studies indicate that late sodium current inhibitor is effective in preventing the increased intracellular calcium-mediated arrhythmias, including long QT syndrome 8. The objective of the study was to determine the role of late sodium current in the calcium related ventricular arrhythmias, L-type calcium channel activator Bay-K 8644 will be used to increase the intracellular calcium concentration.
Methods: Hearts from New Zealand White female rabbits weighing 2.5-3.5 kg were isolated, perfused in a Langendorff mode with modified Krebs-Hensileit solution. The atrioventricular nodal area was thermally ablated to produce complete atrioventricular block, and then heart was paced at stated frequency. Multiple channel monophasic action potentials (MAP) and pseudo 12-lead electrocardiograms (ECGs) were recorded.
Results: Bay-K 8644 (10-300 nM) increased both epicardial and endocardial $MAPD_{90}$ of left ventricle in concentration dependent manners, from (176±6) to (222±13) ms, and (201±6) to (246±10) ms (n=15, P<0.05 vs control), respectively. In the presence of ATX-II, Bay-K 8644 caused greater prolongation of $MAPD_{90}$. Epi-$MAPD_{90}$ was increased from (182±6) to (342±21) ms (n=9, P<0.05 vs control). The prolongation of $MAPD_{90}$ caused by Bay-K 8644 was reversed by 1 M TTX in both absence and presence of ATX-II. In addition, the incidence of PVT evoked by Bay-K 8644 was also related to ATX-II. Bay-K 8644 at the concentration of 200 nM caused few arrhythmias in absence of ATX-II. In contrast, PVT occurred in 7/9 (77.78%) of hearts treated with 200 nM Bay-K 8644 in the presence of ATX-II. These arrhythmias could be abolished by 1 M TTX in the continued presence of Bay-K 8644.
Conclusions: Late sodium current contributes to the intracellular calcium-related ventricular arrhythmias. Inhibition of late sodium current may be effective in preventing or treating calcium overload-related ventricular arrhythmias.

GW25-e5325

The Expression of Stromal Interaction Molecule in Human Mesenteric Artery from Han Chinese Patients with Hypertension

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Objectives: Store operated calcium entry (SOCE) has been shown to contribute to the rise in intracellular calcium concentration ([Ca (2+)] (i)) and associate with artery smooth muscle contraction. Stromal interaction molecule (STIM) is an essential member in SOCE. Whether it is affected in essential hypertension is presently unknown. The aim of this study was to measure the mRNA expression levels of STIM1 and STIM2 in hypertension (HT) and normal arterial pressure (NT) patients.
Methods: Mesenteric arterial tissues were collected from the removed tissues by abdominal operations in Han Chinese normotensive and primary hypertensive patients. The human tissue collection protocol was approved by the Ethics Committee of Luzhou medical College. In the present study, informed consent was obtained from the patients for the use of vascular tissue (which is usually discarded). Mesentery artery mRNA was obtained from 10 NT and 10 HT patients. The expression of mRNA was detected using quantitative Real-time PCR, and the date was analyzed with $2^{-\Delta\Delta CT}$ method. The statistical analysis was done using independent-samples T test by SPSS 17.0.
Results: Average age of HT was 58.37±1.23, while it was 57.63±28.73 in NT. The STIM1 mRNA in hypertension patients showed an increasing trend compared with normal arterial pressure patients, but the difference was not significant (P>0.05). The STIM2 mRNA was significantly increased compared with normal arterial pressure patients (P<0.05). The expression level of STIM2 in HT was three times higher than those in NT.
Conclusions: The expression of STIM2 mRNA was significantly increased in essential hypertension patients. The increase of STIM mRNA expression level is likely to cause functional up-regulation in hypertension patients.

GW25-e0754

Neurohumoral stimulation at peripheral atrial sites: Effects on heart rate and blood pressure

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Objectives: Previous studies have developed the concept of a sympatho-sympathetic reflex (SSR) affecting heart rate (HR) and blood pressure (BP) under pathophysiological conditions. More recently, renal sympathetic denervation (RSD) has been shown to mitigate afferent and efferent sympathetic nerve activity affecting HR and systolic (SBP) / diastolic pressure (DBP). In order to find other sites involved in SSR, applied an adrenergic agent to the right and left atrial appendages (RAA, LAA).
Methods: In 16 pentobarbital anesthetized dogs, a right and left thoracotomy allowed exposure of the RAA and LAA. ECG and blood pressure were continuously monitored. A polyethylene tube was attached across the AAs to form a leakproof barrier. A gauze pad moistened with epinephrine (EPI), 1X10-3, was placed on the AAs after baseline HR and BP were measured.
Results: Within 2 minutes of EPI application to the RAA (n=16) the mean HR increased from 162±24, control to 223±23, EPI P<0.0001; BP increased from a mean of 141/108, control to 189/132, EPI, P<0.01. EPI application to the LAA increased BP from 132/96, control to 194/138, EPI, P<0.001; whereas, there was no significant increase in heart rate, 153±20, control to 157±18, EPI, P>0.05.
Conclusions: Putative stimulation of the autonomic nerves at the RAA and LAA with the adrenergic neurohumor EPI caused differential effects on HR and BP. These findings suggest that afferent information from visceral as well as vascular sites can significantly affect cardiovascular functions.

GW25-e3235

MicroRNA-16 represses myocardial hypertrophy by inhibiting CCND1, CCND2 and CCNE1 expression in cardiomyocytes

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Objectives: Cell-cycle regulatory proteins and microRNAs (miRs) play important roles in cardiomyocyte hypertrophy, but it remains largely unknown how both coordinate during cardiac hypertrophy. Here we investigated whether miR-16 modulates cardiomyocyte hypertrophy by decreasing the expression of cell-cycle regulatory proteins.
Methods: A rat model of pressure-overload hypertrophy induced by abdominal aortic constriction (AAC), a mouse model of pressure-overload hypertrophy induced by thoracic aortic constriction (TAC) and a mouse model of hypertrophy by subcutaneous injection of phenylephrine (PE) were established. Neonatal rat ventricular cardiomyocytes were incubated with PE and neonatal mouse ventricular cardiomyocytes were incubated with Ang-II to induce the hypertrophic phenotype. Quantitative real-time PCR (Q-PCR) was used to detect miR-16 expression, Q-PCR and Western-blot assays were used to determine concerned genes expression. EU staining and phalloidin staining were performed to assess the hypertrophic phenotype of cultured ventricular cells.
Results: We demonstrated that miR-16 expression was markedly decreased in hypertrophic myocardium and hypertrophic cardiomyocytes in rats and mice. Overexpression of miR-16 reduced rat cardiac hypertrophy and hypertrophic phenotype of cultured cardiomyocytes with decreased phosphorylated retinoblastoma (pRb). Conversely, inhibition of miR-16 induced a hypertrophic phenotype. Expression of cyclin (CCN) D1, CCND2 and CCNE1, which can be targeted by miR-16 according to bioinformatic analysis, was up-regulated in rat hypertrophic myocardium and hypertrophic cardiomyocytes. Knockdown of CCND1, CCND2 or CCNE1 inhibited the hypertrophic phenotype with reduced pRb. Furthermore, the signal transducer and activator of transcription-3 (STAT3) and c-Myc were activated during myocardial hypertrophy, which inhibition prevented miR-16 downregulation.
Conclusions: miR-16 inhibits hypertrophy of cardiomyocytes through down-regulation of CCND1, CCND2 and CCNE1 and subsequent cell cycle arrest, suggesting that miR-16 might be a target to manage cardiac hypertrophy.

GW25-e0506

Effects of ischemic postconditioning on sizes of myocardial infarction induced by ischemia/reperfusion and TollR4 expression in rats

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Objectives: To observe the effects of ischemic postconditioning on the sizes of myocardial infarction induced by ischemia/reperfusion (I/R) and expression of Toll-R4 in rats myocardium.
Methods: 48 rats were divided into four groups equally and randomly. Sham group: exposed the heart and without ligation of anterior descending (LAD) branch of left coronary artery; I/R group: Ligated the LAD for 30 minutes, later reperfusion 60 minutes. IPC group: ligated LAD to provoke the myocardial ischemia 30 minutes Firstly and reperfusion for 60 minutes later. IPOC group: Ligated the LAD 30 minutes, and repeating the 1 minute/1 minute ischemia/reperfusion for 3 cycles, then reperfusion 60 minutes. After all the processes above, removed the hearts of the rats, the infarction sizes (IS) were measured with NBT dye, and the expression of Toll-R4 in myocardial tissue was investigated by immunohistochemistry method.
Results: Compared with the I/R group, the IS was reduced significantly (P<0.05), and the expression of Toll-R4 increased significantly in IPOC and IPC groups (P<0.05). There was no significant difference between IPOC and IPC groups.
Conclusions: The postconditioning is as effectivness as preconditioning in reducing infarct size and the expression of Toll-R4 may be one of the mechanisms in reducing the infarction sizes.